V. CLAIMS

What is claimed is:

1. A method for inhibiting cancer cell proliferation comprising administering a NF-κB inhibitor to a subject, wherein the NF-κB inhibitor causes an NFkB inhibition, wherein the subject has cancer cells which are proliferating, wherein the cancer cells are not myeloma cells.

2 A method of promoting cancer cell apoptosis comprising administering a NF-κB inhibitor to a subject, wherein the NF-κB inhibitor causes an NF-κB inhibition, wherein the subject has cancer cells, wherein the cancer cells are not myeloma.

3. A method of inhibiting readhesion of cancer cells to a surface comprising administering a NF-κB inhibitor to a subject, wherein the NF-κB inhibitor causes an NF-κB inhibition, wherein the subject has cancer cells.

4. A method of inhibiting metastasis of cancer cells comprising administering a NF-κB inhibitor to a subject, wherein the NF-κB inhibitor causes an NF-κB inhibition, wherein the subject has cancer cells.

5. The method of claim 4, wherein the NF-κB inhibtor inhibits intraabdominal metastasis.

6. The method of claim 4, wherein the NF-κB inhibitor inhibits hepatic, parietal or peritoneal metastasis.

7. A method of inhibiting tumorigenesis comprising administering a NF-κB inhibitor to a subject, wherein the NF-κB inhibitor causes an NF-κB inhibition, wherein the subject has cancer cells.

8. The method of any one of claims 1-4, wherein the cancer is an abdominal cancer, hepatic cancer, peritoneal cancer, parietal cancer, rectal cancer, stomach cancer, or colon cancer.

9. The method of any one of claims 1-4 or 7, wherein the cancer cells utilize NF-κB for mitogenesis.

10. The method of any one of claims 1-4 or 7, wherein the cancer cells utilize NF-κB for readhesion

11. The method of any one of claims 1-4 or 7, wherein the cancer cell comprises an

APC mutation.

12. The method of any one of claims 1-4 or 7, wherein the cancer cell does not contain an activating mutation on ß-catenin.

13. The method of any one of claims 1-4 or 7, wherein the cancer cell expresses the COX2 gene.

14. The method of claim 12, wherein the cancer cell overexpresses the COX2 gene.

15. The method of claims 1-4, or 7, wherein the cancer cell does not express the COX2 gene.

16. The method of any one of claims 1-4 or 7, wherein the cancer cell is related to a cancer cell line.

17. The method of claim 14, wherein the cancer cell line is a DLD-1 cell line or a HT-29 cell line.

18. The method of any one of claims 1-4 or 7, wherein the cancer cells are colon cancer cells.

19. The method of any one of claims 1-4 or 7, wherein the cancer cells are rectal cancer cells.

20. The method of any one of claims 1-4 or 7, wherein the cancer cells are not adenocarcinoma cells.

21. The method of claim 1, wherein inhibiting cancer cell proliferation is independent of TNFα activated apoptosis.

22. The method of claim 2, wherein promoting cancer cell apoptosis is independent of TNFα activated apoptosis.

23. The method of claim 3, wherein inhibiting readhesion of cancer cells to a surface is independent of TNFα activated apoptosis.

24. The method of claim 4, wherein inhibiting metastasis of cancer cells is independent of TNFα activated apoptosis.

25. The method of claim 7, wherein inhibiting tumorigenesis is independent of TNFα activated apoptosis.

26. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor causes a decrease in the expression of anti-apoptotic proteins.

27. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor inhibits IκB phosphorylation.

28. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor inhibits TNFα induced NF-κB activation.

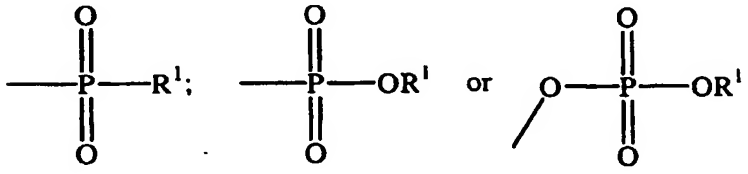
29. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is an olefin.

30. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is an olefin having at least oneelectron-withdrawing group.

31. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is an olefin having at least two electron-withdrawing groups.

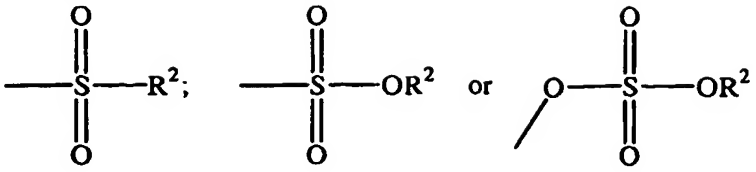
32. The method of claim 29, wherein the electron-withdrawing group comprises a cyano group, a sulfo-oxy group, a phospho-oxy group, a carboxyl group, a nitro group, a halogen, a halogenated alkyl group, an unsubstituted aromatic ring, or a substituted aromatic ring having at least one cyano group, sulfo-oxy group, phospho-oxy group, carboxyl group, hydroxyl group, amino group, ether group, halogenated alkyl group, halogen, or nitro group.

33. The method of claim 31, wherein the phospho-oxy group has the structure



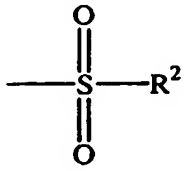
wherein R^1 is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubsituted aromatic. [N&R will define each of these terms in the specification.]

34. The method of claim 31, wherein the sulfo-oxy group has the structure



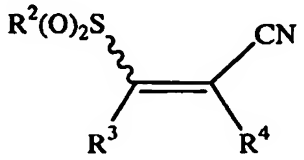
wherein R^2 is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubsituted aromatic.

35. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is an olefin having a cyano group and a sulfo-oxy group having the structure



wherein R^2 is hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubsituted aromatic.

36. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



wherein R^2, R^3 and R^4 are, independently, hydrogen, alkyl, halogenated alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubsituted aromatic, wherein the compound is the E- or Z-isomer.

37. The method of claim 35, wherein R^3 and R^4 are hydrogen.

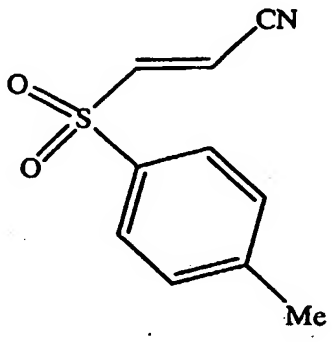
38. The method of claim 35, wherein R^2 is methyl, ethyl, propyl, isopropyl, butyl, isobutyl, tertiary butyl, substituted or unsubstituted phenyl, or benzyl.

39. The method of claim 35, wherein R^2 is a phenyl group having at least one alkyl

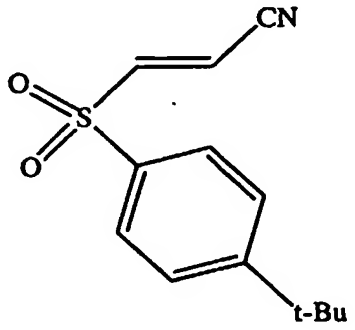
group.

40. The method of claim 35, wherein the compound is the E-isomer.

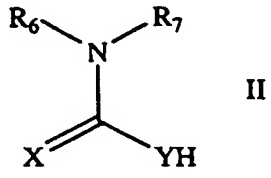
41. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



42. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



43. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has the structure



II

wherein R_6 and R_7 are, independently, hydrogen, alkyl, alkenyl, alkynyl, aralkyl, or substituted or unsubstituted aromatic, or R_6 and R_7 together form a ring with the nitrogen atom,

X and Y are, independently, oxygen or sulfur, or

the pharmaceutically-acceptable salt, ester, or amide thereof.

44. The method of claim 43, wherein X and Y are sulfur, and R_6 and R_7 is $(CH_2)_4$.

45. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor comprises at least one amino acid residue.

46. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor has at least one leucine residue.

47. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor comprises three leucine residues.

48. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is N-[(phenylmethoxy)carbonyl]-L-leucyl-N-[(1S)-1-formyl-3-methylbutyl]-L-leucinamide.

49. The method of claim 40, wherein the NF-κB inhibitor is BAY-11-7082.

50. The method of claim 40, wherein the NF-κB inhibitor is BAY-11-7085.

51. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is MG-132.

52. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor is PDTC.

53. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor directly inhibits NF-κB.

54. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor indirectly inhibits NF-κB.

55. The method of 52, wherein the NF-κB inhibitor inhibits expression of NF-κB.

56. The method of 52, wherein the NF-κB inhibitor inhibits translation of NF-κB.

57. The method of any one of claims 1-4 or 7, wherein the NF-κB inhibitor inhibits NF-κB transport into the nucleus.

58. A method of inhibiting cancer cell proliferation in a subject, comprising testing

for an adenomatous polyposis coli (APC) gene mutation, and if the mutation is detected, administering an effective amount of an NF-κB inhibitor to the subject.

59. The method of claim 58, wherein the NF-κB inhibitor comprises BAY 11-7085.

60. The method of claim 58, wherein the NF-κB inhibitor comprises BAY 11-7082.

61. A method of inhibiting cancer cell proliferation in a subject comprising testing the subject for COX2 expression, and if there was COX 2 expression, administering an NF-κB inhibitor to the subject.

62. The method of claim 61, wherein the NF-κB inhibitor comprises BAY 11-7085.

63. The method of claim 61, wherein the NF-κB inhibitor comprises BAY 11-7082.

64. A method of inhibiting cancer cell proliferation in a subject comprising administering an NF-κB inhibitor to the subject, wherein the subject has had a tumor resected.

65. The method of claim 64, wherein the NF-κB inhibitor is administered prior to the resection.

66. The method of claim 64, wherein the NF-κB inhibitor is administered prior to the resection.

67. The method of claim 64, wherein the NF-κB inhibitor is administered within 10 days of the resection.

68. The method of claim 64, wherein the NF-κB inhibitor is administered within 5 days of the resection.

69. The method of claim 64, wherein the NF-κB inhibitor is administered within 1 days of the resection.

70. The method of claim 64, wherein the NF-κB inhibitor is administered within 10 hours of the resection.

71. The method of claim 64, wherein the NF-κB inhibitor is administered within 1 hour of the resection.

72. The method of claim 64, wherein the NF-κB inhibitor is administered within 0.5 hours of the resection.